

# Neurological symptoms as the result of enlarged dimensions and non-typical course of inferior superficial temporal vein

Ryszard Maciejewski<sup>1</sup>, Andrzej Drop<sup>2</sup>

<sup>1</sup>Department of Human Anatomy, Medical University of Lublin, Poland

<sup>2</sup>Department of Radiology, Medical University of Lublin, Poland

[Received 19 December 2002; Revised 22 January 2002; Accepted 23 January 2002]

*Neurological symptoms as the result of non-typical course of superficial cerebral veins are described in available literature very rarely. The case described below indicates that in some circumstances the compression symptoms derived from the cerebral cortex may be incredibly more serious than their anatomical reasons. In our observation a young woman was described complaining of paroxysmal numbness of the left upper limb with paraesthesiae of the left side of the face, the left eye and left half of the tongue. The patient said that in childhood she used to have paroxysmal itching of the left hand. She also said that CT of the head made a few years ago after a car accident was without pathological changes. Neurological examination, x-ray of the skull and EEG test performed during first visit proved normal. After one year of the disease, Jackson-type epilepsy, combined with loss of sensation of the left half of the face for the first time, was present. Neurological and ophthalmological examination of the bottom of the eye proved normal. Skull x-ray was normal. Then disturbances of the vision in the left half of the field appeared. EEG was still in norm. The MRI test showed the asymmetry in the course and dilated superficial vein between the basis of the right temporal lobe and the tentorium of the cerebellum. The diameter of this vein was 2.5 mm, but there were no vascular malformations. Bottom of the eye was normal, but in the field of vision the white and red colours were dominated.*

**key words:** superficial temporal vein, cerebral venous anomalies, neurological manifestations

## INTRODUCTION

In traditional morphology greater interest was directed to the anatomy of the arterial system of the brain than to the venous system, despite its importance in neuropathology [1–3, 18, 19].

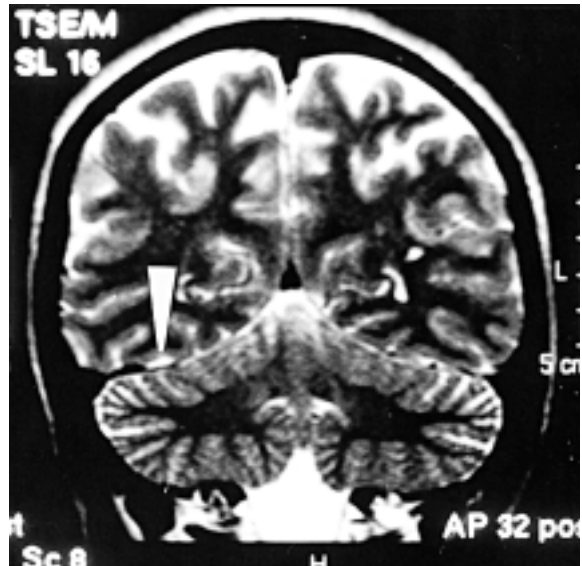
This refers especially to superficial cerebral veins, which may be well developed or may even not exist [9, 13, 14]. Many pathologies can change the normal appearance of the venous system. Knowledge

about normal and developmental anatomy of the venous system may be important in many clinical circumstances. Developmental venous anomalies of the central nervous system are an exaggeration of the normal venous collector system, not a vascular lesion. These anomalies may be associated with other processes with the nervous system like tumours, demyelinating and vascular lesions, as well as cavernous and arteriovenous malformations [16]. The

instigating factors involved are not completely understood. The primary clinical significance of venous anomalies is that planned or inadvertent occlusion during treatment of associated lesions frequently leads to venous infarction of the surrounding normal brain [8]. Recently, increasing attention has focused on the possible importance of venous outflow disturbance and venous hypertension in the pathogenesis and pathophysiology of these anomalies [12]. To our knowledge the first report about the CT angiographic appearance of developmental venous anomalies was published in 1999 [15]. Later other similar reports were presented.

### CASE REPORT

Young woman (20 years — case history 823/99), reported to the Neurological Clinic in October 1999 complaining of paroxysmal numbness of the left upper limb with paraesthesiae to the left side of the face, to the left eye and to left half of the tongue. Paroxysms had increasing intensification with duration in time from 2 to 5 minutes. The patient said that in childhood she used to have paroxysmal itching of the left hand. She also said that CT of the head made in 1994 after a car accident was without pathological changes. Neurological examination, x-ray of the skull and EEG test performed during first visit proved normal. From this time the natural case history was as follows: July 2000 — Jackson-type epilepsy appeared beginning as anaesthesia of the skin around left half of the mouth. Usually it was combined with loss of sensation of the left half of the face. Neurological and ophthalmological examination of the bottom of the eye proved normal. The skull x-ray was without any pathological changes. In October 2000 disturbances of the vision in the left half of the vision field added. In the remaining field of the vision the white and red colours predominated. EEG proved normal. The MRI test showed the asymmetry in the course and dilated dimensions of superficial vein on the inferior surface of the right temporal lobe, above the lateral part of the tentorium of the cerebellum (Fig. 1, 2). The diameter of this vein was 2.5 mm, but there were no vascular malformations. Bottom of the eye was normal, but in the field of vision the white and red colours were still dominant. Then neuralgia of the first branch of the left trigeminal nerve appeared as the main clinical manifestation. Doppler ultrasound examination performed recently showed no vascular malformation in intracranial cavity.



**Figure 1.** MRI a-p scan. Arrow indicates the enlarged superficial vein between the base of the right temporal lobe and tentorium of the cerebellum.



**Figure 2.** MRI sagittal scan. No more vascular malformations are visible from this projection.

### DISCUSSION

Classic description of the superficial cerebral venous system divides these vessels into superior and inferior groups, anastomotic vessels and basal vein. In an anatomical study Kędzia divided the venous system of the lateral cerebral sulcus according to the main direction of the venous outflow [6]. During the past five years, only a few papers have been presented on variations of the superficial venous sys-

tem of the temporal lobe [4, 5, 17]. Four distinct venous drainage complexes were identified on the inferolateral surface of temporal lobe by Guppy et al. [5]. There were: a/ the lateral complex, b/ the anteroinferior complex, c/ the inferiomedial complex and d/ the posteroinferior complex. Three main configurations were described: the candelabra of veins uniting to form one large vein, multiple independent draining veins and venous lakes running in the tentorium before entering the sinuses. The lateral complex was described in 100% of the specimens, but in the majority of cases it did not represent the dominant venous drainage of the lateral and inferior surfaces of the temporal lobe. According to Sener's information, the lateral temporal vein was present on both sides in 80% of examined cadavers, and seen on one or both sides in 0.5% of the CT studies, and in 24% of those using MRI [17]. A search for this vein was undertaken in 20 cadavers, 200 CT and 200 MRI studies [17].

Chen described in the year 2000 a 30-year-old woman with congenital cerebral venous dysgenesis with absence of the internal cerebral and basal veins and most of the cortical veins [4]. Unlike the more common bilateral picture, she had this abnormality only on the left side, which delayed presentation and gave relatively mild symptoms [4]. Topper et al. identified 67 patients with intracranial developmental venous anomalies, among them 12 with associated cavernoma [20]. None of the 67 patients showed an association between the complaints that led to the MRI and the location of the venous anomalies. Authors concluded that these anomalies do not seem to be associated with a specific clinical presentation. The natural history of cerebral venous malformations was well documented by Naff et al. [11]. They stated that the most frequent locations of lesion included the frontal lobe (55.6%) and the cerebellum (27%). The most frequent presentations included headache (50.8%), focal neurological deficits (40%) and seizure (30.2%). Prevalence of headache and seizure decreased over time without treatment of the lesion. Their study established that the natural history of venous malformations is benign and that conservative therapy is warranted. Similar research was done by McLaughlin et al. [10]. They took a 10-year prospective clinical and magnetic resonance imaging study to determine the natural history of 80 patients with cerebral venous malformations. Twenty-two patients presented with neurological signs or symptoms that were thought to be related to the malformations (9 with headaches, 4 with seizures, 3 with

sensory symptoms, 3 with motor deficits, 2 with trigeminal neuralgia). Trigeminal neuralgia caused by compression from petrosal vein transfixing the nerve was also described by Kimura et al. [7]. All the above presented cases indicate that an understanding of the complexity and diversity of the intracranial venous drainage complexes and their configurations is necessary to avoid diagnostic and therapeutic problems. It should simplify the decision about final operative or non-operative treatment in the future.

## REFERENCES

1. Ciszek B, Ząbek M (1992) Anatomia tętnicy środkowej mózgu a obraz angiograficzny jej tętniaków. *Neurol Neurochir Pol, Suppl 1*: 67–72.
2. Ciszek B, Ząbek M (1992) Rzadkie odmiany tętnicy środkowej mózgu. *Neurol Neurochir Pol, Suppl 1*: 72–82.
3. Ciszek B, Dąbrowska M, Andrzejczak A, Aleksandrowicz R, Mazurowski W, Ząbek M (1998) Middle superficial cerebral vein. *Folia Morphol (Warsz.)*, 57: 149–155.
4. Chen CJ (2000) Unilateral congenital cerebral venous dysgenesis. *Neuroradiology*, 42: 40–42.
5. Guppy KH, Origiano TC, Reichman OH, Segal S (1997) Venous drainage of the inferolateral temporal lobe in relationship to transtemporal/transtentorial approaches to the cranial base. *Neurosurgery*, 41: 615–619.
6. Kędzia A (1974) Zlewisko żylne dołu i bruzdy bocznej mózgu. *Neuropat Pol*, 12: 515–530.
7. Kimura T, Sako K, Tohyama Y, Yonemasu Y (1999) Trigeminal neuralgia caused by compression from petrosal vein transfixing the nerve. *Acta Neurochir (Wien)*, 141: 437–438.
8. Maeder P, Gudinchet F, Meuli R, deTribollet N (1998) Development of a cavernous malformation of the brain. *Am J Neuroradiol*, 19: 1141–1143.
9. McKinnon SG (1998) Anatomy of the cerebral veins, dural sinuses, sella, meninges, and CSF spaces. *Neuroimaging Clin N Am*, 8: 101–117.
10. McLaughlin MR, Kondziołka D, Flickinger JC, Lunsford S, Lunsford LD (1998) The prospective natural history of cerebral venous malformations. *Neurosurgery*, 43: 195–200.
11. Naff NJ, Wemmer J, Hoenig-Rigamonti K, Rigamonti DR (1998) A longitudinal study of patients with venous malformations: documentation of a negligible hemorrhage risk and benign natural history. *Neurology*, 50: 1709–1714.
12. Nussbaum ES, Heros RC, Madison MT, Awasthi D, Truwit CL (1998) The pathogenesis of arteriovenous malformations: insights provided by a case of multiple arteriovenous malformations developing in relation to a developmental venous anomaly. *Neurosurgery*, 43: 347–351.
13. Oka K, Rhoton AL, Barry M, Rodrigues R (1985) Microsurgical anatomy of the superficial veins of the cerebrum. *Neurosurgery*, 17: 711–748.

14. Parese DM (1960) Superficial veins of the brain from a surgical point of view. *J Neurosurg*, 17: 402–412.
15. Peebles TR, Vieco PR (1997) Intracranial developmental venous anomalies: diagnosis using CT angiography. *J Comput Assist Tomogr*, 21: 582–586.
16. Pryor J, Setton A, Berenstein A (1999) Venous anomalies and associated lesions. *Neurosurg Clin N Am*, 10: 519–525.
17. Sener RN (1996) The lateral temporal vein: a cadaver, CT and MRI study. *Neuroradiology*, Suppl 1: 57–59.
18. Stephens RB, Stilwell DL (1969) Arteries and veins of the human brain. Charles C Thomas, Springfield IL.
19. Taveras JM, Wood EH (1976) Diagnostic neuroradiology. Williams & Wilkins, Baltimore, Vol. 2, Part III.
20. Topper J, Jurgens E, Reul J, Thron A (1999) Clinical significance of intracranial developmental venous anomalies. *J Neurol Neurosurg Psychiatry*, 67: 234–238.